Recombinant Mouse Resistin

Catalog Number: 5335-RN

DESCRIPTION

Source	Mouse myeloma cell line, NS0-derived Ser21-Ser114 Accession # Q99P87
N-terminal Sequence Analysis	Ser21
Structure / Form	Homohexamer
Predicted Molecular Mass	10.2 kDa

SPECIFICATIONS

SDS-PAGE	10 kDa, under reducing conditions
Activity	Measured in a cell proliferation assay using HUVEC human umbilical vein endothelial cells. The ED_{50} for this effect is 0.8-4 µg/mL.
Endotoxin Level	<0.01 EU per 1 µg of the protein by the LAL method.
Purity	>95%, by SDS-PAGE under reducing conditions and visualized by silver stain.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 500 µg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	**Use a manual defrost freezer and avoid repeated freeze-thaw cycles.** ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 3 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

Resistin (resistance-to-insulin), also known as adipocyte-specific secretory factor (ADSF) and found in inflammatory zone 3 (FIZZ3), is a 10 kDa member of a small family of secreted cysteine-rich peptide hormones. These molecules purportedly play some role in inflammation, glucose metabolism, and angiogenesis (1, 2, 3, 4). Mouse Resistin precursor is 114 amino acids (aa) in length. It contains a 20 aa signal sequence plus a 94 aa mature region. The mature region shows an N-terminal α-helical tail (aa 27 - 48) and a C-terminal β-sheet globular head (aa 51 - 112) (5, 6). The Resistin molecule circulates as either a noncovalent trimer (minor form), or a disulfide-linked homohexamer (major form). Noncovalent trimers are generated when the α-helical segments self-associate to form a three-stranded coiled-coil structure. Covalent hexamers subsequently appear when the free Cys at position #26 is engaged by adjacent trimers. It is hypothesized that the hexamer is a less active form of the molecule, and bioactivity is achieved at the target site by disulfide bond reduction (5). Although Resistin family molecules can noncovalently interact to form heterotrimers *in vitro*, there is no evidence to suggest this occurs *in vivo* with Resistin (7, 8). Mature mouse Resistin shares 72% and 56% aa identity with rat and human Resistin, respectively. Rat Resistin possesses an alternate start site at Met48; this Met is not found in the mouse molecule, however (9). Rodent Resistin is expressed by white adipocytes, splenocytes, astrocytes, and anterior pituitary epithelium (6, 10, 11). Although the function of Resistin is unclear, it would seem to block insulin-stimulated uptake of glucose by adipocytes, and promote glucose release by hepatocytes (6, 12, 13). As such, it has been proposed to participate in diet-induced insulin-sensitivity. Diets high in fat promote an increase in overall adipocyte size. Hypertrophic adipocytes are known to secrete TNF-α, which acts locally to block ACRP30 production. Since ACRP30 is an insulin-sensitizer, a drop in ACRP30 availability leads to insulin-insensitivity, which drives increased insulin production (a compensatory mechanism). High insulin induces Resistin secretion, which now antagonizes insulin action, prompting more insulin production, and more Resistin secretion (14).

References:

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